

What is claimed is:

1. A method of prophylaxis against myocardial infarctions which exhibit CK-MB levels greater than about 50 nano-grams/ml in a subject comprising:

5 administering to the subject undergoing a procedure involving cardiopulmonary bypass an effective myocardial infarction reducing amount of an anti-inflammatory compound.

2. The method of claim 1, wherein the procedure is CABG surgery.

3. The method of claim 1, wherein the CK-MB level is greater than about 60 nano-grams/ml.

10 4. The method of claim 1, wherein the CK-MB level is greater than about 70 nano-grams/ml.

5. The method of claim 1, wherein the CK-MB level is greater than about 80 nano-grams/ml.

15 6. The method of claim 1, wherein the CK-MB level is greater than about 90 nano-grams/ml.

7. The method of claim 1, wherein the CK-MB level is greater than about 100 nano-grams/ml.

8. The method of claim 1, wherein the CK-MB level is greater than about 120 nano-grams/ml.

20 9. The method of claim 1, wherein the anti-inflammatory compound is a complement inhibitor.

10. The method of claim 9, wherein the complement inhibitor is selected from the group consisting of a) antibodies directed against complement components C-1, C-2, C-3, C-4, C-5, C-6, C-7, C-8, C-9, Factor D, Factor B, Factor P, MBL, MASP-1, or MASP-2; and b) naturally occurring or soluble forms of CR1, LEX-CR1, MCP, DAF, CD59,
5 Factor H, cobra venom factor, FUT-175, y bind protein, complestatin, or K76COOH 2.

11. The method of claim 10, wherein the antibody directly or indirectly reduces the conversion of complement component C5 into complement components C5a and C5b.

10 12. The method of claim 11, wherein the anti-C5 antibody is an antibody comprising at least one antibody-antigen binding site, said antibody exhibiting specific binding to human complement component C5, said specific binding being targeted to the alpha chain of human complement component C5, wherein the antibody 1) inhibits complement activation in a human body fluid; 2) inhibits the binding of purified human complement component C5 to either human complement component C3 or human complement component C4 ; and 3) does not specifically bind to the human
15 complement activation product for C5a.

13. The method of claim 9, wherein the complement inhibitor specifically binds to a component forming the C5b-9 complex.

14. The method of determining effectiveness of an anti-inflammatory compound in reducing incidence of myocardial infarction comprising:

20 administering the compound to a subject group comprising at least one patient undergoing a procedure involving cardiopulmonary bypass; and

comparing incidence of infarctions in the subject group to incidence of infarctions in a control sample of patients when the peak level of CK-MB in the blood is greater than 50 nano-grams/ml in both groups;

wherein a decrease in the incidence of infarctions in the subject group indicates effectiveness of the compound.

15. The method of claim 14, wherein the procedure is CABG surgery.

16. The method of claim 14, wherein the CK-MB level is greater than about 60 nano-grams/ml.

17. The method of claim 14, wherein the CK-MB level is greater than about 70 nano-grams/ml.

18. The method of claim 14, wherein the CK-MB level is greater than about 80 nano-grams/ml.

19. The method of claim 14, wherein the CK-MB level is greater than about 90 nano-grams/ml.

20. The method of claim 14, wherein the CK-MB level is greater than about 100 nano-grams/ml.

21. The method of claim 14, wherein the CK-MB level is greater than about 120 nano-grams/ml.

22. The method of claim 14, wherein the anti-inflammatory compound is a complement inhibitor.

23. The method of claim 22, wherein the complement inhibitor is selected from the group consisting of a) antibodies directed against complement components C-1, C-2, C-3, C-4, C-5, C-6, C-7, C-8, C-9, Factor D, Factor B, Factor P, MBL, MASP-1, or MASP-

